Unexpected outcome (positive or negative) including adverse drug reactions

Conservative management in a rare case of spontaneous bilateral cerebellar haemorrhage

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Summary

Intracranial haemorrhage is usually associated with various risk factors such as hypertension, aneurysm, bleeding diatheses, anticoagulant use, amyloid angiopathy and remote bleed occurring after supratentorial and spinal surgery. Simultaneous bilateral cerebellar haemorrhage is rarely observed outside the setting of known precipitants and may follow a rapid downhill course. We present an unusual case of a young man who presented to us with sudden-onset cerebellar signs due to haemorrhage occurring in both the cerebellar hemispheres. Our patient had a favourable outcome with conservative management despite having bilateral cerebellar haematoma with partial obliteration of the fourth ventricle.

BACKGROUND

Intracranial haemorrhage (ICH) is detected in almost 10% of all cases of stroke. The most common site for ICH is the putamen (35%) followed by lobar and thalamic haemorrhage. Unilateral cerebellar haemorrhage accounts for 5–10% of the cases of intracerebral haemorrhage¹; however, the occurrence of bilateral cerebellar haemorrhage is rare. We herein report this rare occurrence in a young man in the absence of any known predisposing risk factors.

CASE PRESENTATION

A 38-year-old right-handed man presented with complaints of sudden-onset giddiness and swaying while walking (left more than right) 1 day before admission. The symptoms progressed within 2–4 h and the patient started swaying even while sitting upright. This was accompanied by throbbing headache that was severe in intensity, started in the occipital region and the nape of the neck and later on became holocranial. The patient became extremely restless and irritable because of the headache and had 8–10 episodes of projectile vomiting. There was no suggestion of convulsions, transient loss of consciousness or focal neurological deficit. There was no history of fever, rash, hypertension, diabetes mellitus, bleeding diathesis, the use of anticoagulants, drug abuse or trauma.

Blood pressure at the time of admission was 128/84 mm Hg, with a Glasgow Coma Scale score of 15/15. Cerebellar signs in the form of hypotonia, dysdiadochokinesis and dysmetria, involving the left side more than the right, were present in addition to truncal and gait ataxia. There was no evidence of nystagmus, cranial nerve palsy, ataxic dysarthria or any other neurological deficit.

INVESTIGATIONS

The routine haemogram, including platelets, and the biochemical parameters of the patient were within normal limits. Estimation of bleeding time, clotting time, international normalised ratio and activated partial thromboplastin time did not reveal any abnormality. Non-structural

protein 1 and IgM to dengue virus were not detected; serum was found to be non-reactive to human immunodeficiency virus I and II, and hepatitis B and C viruses. A CT of the brain showed lanceolate hyperdensities in both the cerebellar hemispheres, measuring 4.14×1.77 cm on the left side and 2.3×1.2 cm on the right side, with partial obliteration of the fourth ventricle; involvement of the subarachnoid space was also noted (figure 1). An MRI of the brain showed blooming on T2W gradient-echo sequence suggestive of acute bilateral cerebellar bleed (left>right) with perilesional oedema and compression of the fourth ventricle (figure 2). Additional angiographic studies (based on MRI as well as CT), done to look for the presence of any aneurysm, arteriovenous malformation or venous thrombosis, were found to be normal.

TREATMENT

In the absence of consent for surgery, the neurosurgical team decided to monitor the patient for the development of any deficit or suggestion of raised intracranial pressure. The patient was, therefore, managed conservatively with intravenous mannitol (0.5 gm/kg 6 hourly), with per-oral acetazolamide (250 mg thrice a day). Parenteral metoclopramide (10 mg) and pantoprazole (40 mg) were used concurrently.

OUTCOME AND FOLLOW-UP

The patient responded well to conservative therapy and after 5 days of the same, did not have any headache or vomiting. Mannitol and acetazolamide were tapered and stopped after 10 days. At 2 weeks, the patient was able to sit without support and feed himself with some assistance from the attendants. A follow-up CT of the brain done at this time revealed partial resolution of the haemorrhage (right>left) involving the cerebellar hemispheres. At 3 weeks, when the patient was discharged, the patient had mild appendicular and gait ataxia.

DISCUSSION

The most common aetiological factor in cerebellar haemorrhage is hypertension.¹ The other causes of cerebellar

Figure 1 Axial sections of non-contrast CT of the brain show bilateral lanceolate haemorrhage (left>right) in the cerebellar hemispheres at the level of mid-to-low mesencephalon (A, B), with a normal-contrast CT angiography (C). There is partial obliteration of the fourth ventricle along with involvement of the subarachnoid space (A, B). A Follow-up CT (D) done at 3 weeks depicts resorption of the haemorrhage, with residual hypodensity, in the earlier involved areas of the cerebellum.

haemorrhage are ruptured aneurysm, arteriovenous malformations, tumour, anticoagulant or thrombolytic therapy, infections (eg, dengue) and drug abuse (eg, cocaine and other sympatho-mimetic drugs).^{1 2} Cerebral amyloid angiopathy is an important but under-recognised cause of ICH in the normotensive elderly.³ It is an unusual cause of cerebellar bleed and has been documented as a coexistent pathology in a young man with arteriovenous malformation.⁴ There have been few case reports regarding spontaneous remote cerebellar bleeds occurring as a result of supratentorial craniotomies and spinal surgery.⁵⁻⁷

Spontaneous bilateral cerebellar haemorrhage is a rare condition. Most of the cases of bilateral cerebellar haemorrhage reported in the literature are due to remote cerebellar haemorrhage (RCH) occurring as a complication after supratentorial craniotomy,⁵ spinal surgery and lumboperitoneal shunt.^{6 7} The probable pathophysiology involved in RCH is tearing of the superior cerebellar vein leading to haemorrhagic venous infarcts. On a CT, streaky curvilinear areas of increased attenuation in the cerebellar sulci

and folia are seen; these appear like stripes of a zebra, and are hence known as the 'zebra sign'.⁸ RCH is a relatively benign condition and does not require aggressive management.⁵

Poorly controlled blood pressure may lead to sequential haemorrhage in the cerebellum leading to bilateral haematoma due to rupture of Charcot-Bouchard microaneurysms.⁹ In the cerebellum, small penetrating branches arising from superior cerebellar arteries are the usual site of such microaneurysms which occur because of the degenerative effect of long-standing hypertension. This could be the reason why most of the haematomata are usually detected in the vascular territory of the superior cerebellar artery.¹⁰ Our patient also had involvement in the similar territory but any vascular abnormality could not be demonstrated.

The clinical manifestation of cerebellar haematoma is that of an abrupt onset, severe vertigo and ataxia in the absence of weakness, along with signs of raised intracranial pressure such as headache, vomiting and drowsiness.

Figure 2 An MRI of the brain shows isointense to hypointense lanceolate lesions involving the cerebellar hemispheres on an axial T1W image (A). The lesions appear hypointense on axial T2W (B), axial fluid-attenuated inversion recovery (C), T2W coronal (F) and T2W sagittal (G) images with hyperintense perilesional oedema. The lesion blooms on T2W gradient-echo image (D) suggestive of a haemorrhage whereas no restriction is evident on a diffusion-weighted image (E). Partial obliteration of the fourth ventricle can be appreciated on the axial sections (A–D). A time-of-flight angiography (H) did not reveal any abnormality.

There may be involvement of ipsilateral cranial nerves in lobar haemorrhage, the abducens and facial nerve being the most common ones to be involved. Involvement of the vermis as a result of an extension of the haematoma or perilesional oedema can cause truncal ataxia.

It is extremely difficult to predict the clinical outcome in cerebellar haemorrhage right at the onset. Even slight expansion of haematoma or increase in perilesional oedema can lead to abrupt deterioration and to coma or death.¹¹ The volume of haemorrhage, Glasgow Coma Scale score on admission and the presence of hydrocephalus have been found to be strong predictors of 30-day mortality.^{12–13} The recent practice guidelines for the management of ICH recommend that patients with cerebellar haemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the haemorrhage as soon as possible. The most important revision to the previous guidelines is deletion of the 3 cm cut-off value for cerebellar haematoma which is no longer considered absolute.¹⁴ Earlier Kirolos *et al*¹⁵ in a non-randomised prospective study had also concluded that in a conscious patient surgical evacuation of cerebellar haematoma which is >3 cm is not required in the absence of fourth ventricle obliteration. Recombinant factor VIIa and vitamin K have been recommended in haemophiliacs sustaining ICH and those with anticoagulant-associated haemorrhage, respectively.¹⁶ Our patient did not have any amenable predisposing factor owing to which a similar therapy was not attempted.

The positive outcome in our patient despite bilateral cerebellar haemorrhages, brainstem compression and early hydrocephalus was very unusual. This suggests that a normal Glasgow Coma Scale score at the time of presentation and an improving course might be a better predictor of good outcome.

Learning points

- ▶ Sudden-onset severe headache, giddiness and ataxia are an ominous triad and warrant urgent neuroimaging.
- ▶ Bilateral cerebellar haemorrhage is a rare condition and precipitants like the presence of an aneurysm, arteriovenous malformation, cortical venous sinus obstruction and bleeding diatheses should always be ruled out.
- ▶ In the absence of complete obliteration of the fourth ventricle, cerebellar haematomas larger than 3 cm may be managed medically.

Competing interests None.

Patient consent No, the patient was not involved in clinical trial.

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